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On Books

The Tangled Tale of Genes and Environment: Moore's *The Dependent Gene: The Fallacy of "Nature vs. Nurture"*

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Nature–nurture views that smack of genetic determinism remain prevalent. Yet, the increasing knowledge base shows ever more clearly that environmental factors and genes form a fully interactional system at all levels. Moore's book covers the major topics of discovery and dispute, including behavior genetics and the twin studies, developmental psychobiology, and developmental systems theory. Knowledge of this larger life-sciences context for behavior principles will become increasingly important as the full complexity of gene–environment relations is revealed. Behavior analysis both contributes to and gains from the larger battle for the recognition of how nature and nurture really work.

Key words: nature–nurture relations, heritability, genetics, evolution, developmental systems theory

Misunderstandings about "nature versus nurture" remain prevalent in biology and psychology as well as in the public sphere. As D. S. Moore (hereafter, Moore) points out in *The Dependent Gene*, contributing to the problem are the common cultural assumptions that (a) genes program for many traits, with the environment in a subordinate role; and that (b) genetic and environmental contributions to a trait can be separated in a sort of percentage game. The true story is more complicated, even for anatomical features and what are called genetic diseases. The crux of the matter is that genes and environment must work together to produce *any* aspect of any living thing. To demonstrate this fact in its glorious complexity, Moore takes readers on a brief historical tour and then tackles heritability and the twin studies, genetics, embryology, neuroscience, gene–environment interactions large and small, developmental psychobiology, evolution, and a sampling of the implications.

Those implications for behavior analysis are profound. Recognition of the full scope of environmental factors requires recognition of the full scope of the behavior principles that behavior analysts study and apply. Behavior principles influence and are influenced by biological and evolutionary processes at all levels, from the molecular to the millennial (e.g., Avital & Jablonka, 2000; Schneider, 2003). The battle against simplistic genetic determinism has rallied behavior analysts since John B. Watson, and continues to concern them deeply. This review focuses on three areas integral to the nature–nurture debate: genes, heritability, and development and evolution.

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GENES

On Genetic Determinism

To begin with the basics, given a "normal" environment, a common assumption is so-called genetic deter-

mination of the number of fingers and toes, say. But alternatively, given a "normal" genome, so-called environmental determination of the number of fingers and toes could be claimed. Consider the case of the teratogen thalidomide, which frequently altered this number during a tragic period in the 20th century. Further, smoking during pregnancy is one of several documented environmental risk factors, although the evidence is correlational (see Man & Chang, 2006, who demonstrated an epidemiological dose–effect relation). Indeed, at an elementary level, a host of the right environmental factors must be present at the right times and in the right places. Both genes and environmental factors are *always* necessarily involved.

Obviously, this conclusion in no way diminishes the importance of the study of genetic contributions. Recent advances in genetics have been critical in demonstrating the often Byzantine ways in which multiple genes and multiple environmental factors interact. But headlines proclaiming discoveries of the "gene for" a wide range of human characteristics, including personality and other behavioral traits, require clarification in a number of respects.

First, a fundamental principle was well characterized in the early history of genetics. As noted in *The Dependent Gene*, Sturtevant pointed out at the beginning of the 20th century that, although a single gene had been found to be responsible for a *difference* in fruit fly eye color, other factors being held as equal as possible, that gene in no sense could be taken to code *for* eye color. Instead, eye color was the result of many genes and many environmental factors. Moore suggests as an analogy the necessity of wheels and a chain in order for bicycle pedals to operate *for* forward motion.

Second, even with this important proviso, the simple single-gene single-trait systems popular in the media are rare—and more complex than they seem. Consider the small number of genetic diseases that are classified as monogenic.[1] Deriving from an abnormality in a single gene, these constitute a very small fraction of diseases (Jablonka & Lamb, 2005). In those monogenic diseases considered to be autosomal recessive (i.e., Mendelian and not on the X or Y chromosome), recessive homozygosity (two copies of the recessive form) does not necessarily result in the problematic phenotype. Nope. Put technically, although those at genetic risk can be highly likely to develop the disease, the penetrance is never 100%, and it is sometimes considerably lower (see Morange, 2001, for examples and commentary). Phenylketonuria (PKU), a classic genetic disease of this type that is discussed in *The Dependent Gene*, is characteristic in that the severity varies despite the same homozygosity—even controlling for exposure to the problematic amino acid that cannot be metabolized. "The evolution towards seeing single-gene traits as versions of complex traits has been under way for some time," noted Scriver and Waters (1999, p. 267). They summarized numerous reasons for the variability in PKU outcomes, finding that the major gene was just one of many factors; indeed, "the whole organismal phenotype is more than the sum of the parts; it is an emergent property" (p. 272). The Centers for Disease Control's panel of experts concluded in a general statement that, "As we acquire more knowledge about the molecular basis of genetic disease, it becomes increasingly clear that variable expressivity (i.e., modification of a genetic trait by other genes or the environment) is the rule

[1] Note the categorization difficulties with respect to the fuzzy set of nonmonogenic genetic diseases; note also other complications, such as the fact that the problematic allele for the monogenic disease sickle cell anemia is actually beneficial in heterozygous individuals.

rather than the exception'' (Burke et al., 1998, quoted in Moore, p. 230).

Third, in the version known as a *phenocopy*, PKU, like other genetic diseases, can develop in the absence of the known gene form (Gray, 2001; see R. Moore et al., 2001, on Huntington's disease). Sometimes the problematic mechanisms are identical and sometimes they are different, but they result in either identical or nearly identical symptoms. It will not come as a surprise that the same symptoms can be associated with either or both genetic and environmental abnormalities in various combinations. In living systems, there are multiple pathways to the same end.

Finally, a single gene commonly influences many traits (pleiotropy). Morange (2001) concluded, for example, that

> There are no proteins specific to learning and memory but rather proteins that, through their function as relays or transmitters, have been harnessed by evolution in the development of cognitive processes. … What makes a process specific is not the nature of its molecular components (and thus the genes that code for these components) but the way they are used and assembled in particular molecular pathways and specific structures. (pp. 88–89)

These higher level operations necessarily involve environmental factors.

''Genetically determined'' could be seen as useful shorthand in some cases, such as when a genetic feature like single-gene dominance or recessive homozygosity often produces given effects across a large range of ''normal'' environments, just as, vice versa, ''environmentally determined'' could for an environmental feature that often produces given effects across a large range of ''normal'' genomes. But these simplifications can become problematic: They get overgeneralized, and the fact that they are simplifications can be forgotten.

Defining the Gene

The very process by which genes are said to code for proteins is far from simple. Most DNA, including human DNA, is what is known as ''junk''—remnants from the past, a proportion of which has turned out to be regulatory. *Cistrons*, which constitute a tiny proportion of human DNA, are those portions of a chromosome that can actually code for a protein. However, the cistrons are not simple uninterrupted sequences of the relevant nucleotides; instead, they contain *exons* that actually hold the sequence, intermingled with nucleotides that generally appear not to code for anything. Sometimes the exon is a small portion of the cistron. The cellular environment is critical for the selection of the proper nucleotides to read. After all, all cells that constitute an organism contain the same genome in their nuclei. Further, the same cistron can be operated on in different ways to code for different proteins. A substantial proportion of human DNA makes use of such *alternative splicing*. Jablonka and Lamb (2005) noted that one gene in the chicken has been found to have 576 different splice versions—although, as is usually the case, these are minor variations of each other. Finally, after the nucleotides are properly sequenced, the environment has long been recognized as essential for actual protein construction. For example, a protein's shape, usually critical for its function, depends on environmental features as well as on the sequence of specified amino acids.

This collection of findings means that the very definition of a gene can be less than straightforward. Although the generic cistron usually qualifies, as Keller (2000) noted,

> The gene has lost a good deal of both its specificity and its agency. Which protein should a gene make, and under what circumstances? And how does it choose? In fact, it doesn't. Responsibility for this decision lies elsewhere, in the complex regulatory dynamics of the cell as a whole. It is from these regulatory dynamics, and not from the gene itself, that the signal (or signals) determining the specific pattern in which the final tran-

> script is to be formed actually comes. (p. 63, quoted on p. 67 of Jablonka & Lamb, 2005)

In other words, environmental factors are critical in determining what protein-coding exons get read from a cistron, when, and how often. Thus, the very concept of a gene requires the environment. As Moore puts it,

> Such contextual dependence renders untenable the simplistic belief that there are coherent, long-lived entities called "genes" that *dictate* instructions to cellular machinery that merely constructs the body accordingly. The common belief that genes contain context-independent "information"—and so are analogous to "blueprints" or "recipes"—is simply false. (p. 81)

Strictly speaking, then, as Gray (1992) concluded, "a gene can only be functionally defined in a specific developmental context" (cited in Moore, p. 81).

Genes, Epigenetics, and Epigenetic Inheritance

The cellular-level mechanisms involved in these operations are *epigenetic*, meaning that they entail nongenetic factors that are inherited themselves or that affect genetic inheritance and gene expression. For example, DNA methylation, which does not affect the genotype, reduces the likelihood of gene expression. Methylation patterns can themselves be inherited. If the capacity to produce proteins for a needed function is present in the genome, it can be unmasked through epigenetic means. (A dramatic demonstration was Kollar & Fisher's, 1980, induction of teeth from bird tissue in vitro.) The resuscitated gene can then be available once again for natural selection to act on. Epigenetic mechanisms also have large effects on the DNA that helps regulate the protein-coding genes, such as the transposons (so-called *jumping genes* that constitute a large proportion of the mammalian genome). And, as Jablonka and Lamb (2005) noted, epigenetic changes are reversible, thus offering readier adaptability to changing conditions than changes in the genes themselves.

These epigenetic mechanisms are in turn responsive to more molar-level environmental factors. But so of course is the genome itself. Chemicals and electromagnetic emissions are among the environmental factors well known to be capable of altering DNA directly. Environmental and behavioral factors routinely modify gene expression and activity (see Gottlieb, 1997, 1998, for numerous documented ways). *Immediate early genes*, for example, are activated by environmental signals. Genetic mutations even appear to be induced when and where needed to some extent (see Jablonka & Lamb, 2005, for an extensive discussion). Stress is one of the factors documented to result in mutations in the DNA (and some controversy exists over whether that phenomenon has been selected for or is simply a side effect). In any event, as Moore notes, "our daily experience of stress directly impacts the activity of our genes" (p. 139). Compensations for these stress effects can occur by an adjustment of the expression of the same gene or in other ways (e.g., Francis, Diorio, Plotsky, & Meaney, 2002): Again, multiple pathways exist toward the same end.[2] Cellular epigenetic mechanisms are among the important mediators.

Several types of epigenetic inheritance systems have been discovered, including RNA interference (subject of a recent Nobel prize), prions, and DNA methylation and other forms of chromatin marking (i.e., marks on

[2] In the same way, gene knockouts can have surprising effects, sometimes even causing improvements in functioning although the protein coded for had been thought to be critical (e.g., Morange, 2001). In this regard, Jablonka and Lamb (2005) refer to "the dynamic regulatory structure of the network" (p. 63) as a potent force for stability. Moore has been criticized for failing to discuss the knockout gene studies, but they can actually be taken to bolster his case.

the materials that form the chromosomes). As contrasted to the genetic system, the epigenetic inheritance system transmits phenotypes, not genotypes, a feature it shares with behavioral inheritance systems (see below). Further, changes during an organism's own lifetime are sometimes inherited, in Lamarckian fashion, across generations of organisms as well as of cells within an organism (Jablonka & Lamb, 1995, 2005). Astonishingly, one researcher turned some of the cilia of a paramecium inside out—and the change was inherited (Beisson & Sonneborn, 1965, also cited in Jablonka & Lamb, 2005, p. 122). Such environmentally induced changes can sometimes become assimilated in the genome, a fact known since Waddington's classic fruit fly experiments in the 1940s (see Avital & Jablonka, 2000; also see Moore, p. 202). These mechanisms do not act only in single-celled organisms. Pavelka and Koudelova (2001) found that Mediterranean flour moth larvae with a mutation for short antennae developed normal-length antennae as adults, if raised at a higher incubation temperature than normal during a sensitive period. Their offspring for several generations retained this feature, despite the short-antennae genotype, *and* despite being raised at the normal incubation temperature, with epigenetic inheritance mechanisms considered the most likely cause. Examples in vertebrates also exist (e.g., mouse health characteristics and coat color; see Jablonka & Lamb, 2005).

HERITABILITY

Inheritance is complex, and Moore's deconstruction of heritability shows how simplistic and misleading the usage of that construct has often been. *Heritability* is defined as the proportion of trait variation associated with corresponding genetic variation—in a particular population under particular circumstances. Figures like 70% have been produced for the heritability of IQ; the heritability of autism has also received a high estimate. What do these numbers really mean?

Understanding Heritability

To begin, Moore summarizes a famous illustration by Lewontin (1970). If seeds varying in genetic constitution are raised in identical environments, any differences among the plants, such as height, must be due to genetic variation. Thus, heritability for height (or any other trait) must be 100%. If the same seeds are sown in identical hostile environments, all the plants are much shorter, but height differences among them must still be due to genetic variation, and heritability remains 100%. Yet, the differences between these two groups obviously depend on the environments. And, whatever the heritability, plants need soil, water, and sunlight to grow.

Moore continues the analogy with the example of cloned seeds (seeds with identical genes) raised in environments that are not identical. In this case, any height or other trait differences must be due to environmental differences, so heritability is 0. But genes are obviously necessary. For the same trait in the same species, then, heritability can vary throughout its range as a function of circumstances. (Indeed, the heritability of IQ has long been known to be substantially lower in children than in adults, e.g., Block, 1995.)

Two examples drawn by Moore from Block (1995) bring home the point. First, the number of human fingers and toes has very low heritability. Variability in digit number is largely accounted for by accidents or disease—environmental factors, not genetic variation. As discussed previously, the teratogen thalidomide provides one example: When it acts prenatally, despite a "normal" genome, an abnormal number of digits

can result. Second, the wearing of earrings in 1950s America had high heritability: Only females used to be likely to wear earrings then, explaining the genetic correlation. But cultural factors were clearly as critical then as they are now for this behavior, now that its heritability must be lower. So, although heritability *sounds* like it quantifies the degree to which a trait itself is determined by genes, it does not. (And of course it could not: Genes and environmental factors are both always necessary; recall Moore's example of the bicycle pedals *for* forward propulsion only in conjunction with other essential parts.)

Limitations, Confusions, and Confounding Variables

This is just the beginning of the confusions concerning this correlational construct. Heritability estimates statistically apportion sources of variation in traits, but they apply only to the specific populations and contexts from which they are derived. They cannot be generalized to other populations or circumstances without extra empirical evidence. And if the original context varies—if environments are sometimes similar and sometimes different in ways that affect the trait—the estimates themselves are confounded. Heritability estimates apply only to groups, and are inherently inapplicable to individuals in any sense. And they do not imply causation. As Moore notes, all of these important limitations have been frequently ignored or minimized.

Consider also a pair of identical twins reared in different environments. If analogous plant clones grow to the same height in different environments, this identical outcome cannot be concluded to be "programmed by the genes" in any sense: Lack of sun in one location may be matched in effect by poor soil in another, for example. Similarly, identical twins raised in different environments may share a trait outcome not because of their shared genes, but because of similar *or* different features of their different environments, features that might have produced the same outcome regardless of a wide variety of genetic differences. Along these lines, being raised in the same family does not mean that environments do not vary in many significant ways. Just one such difference can be enough to create a large and long-lasting effect on a trait, a point John B. Watson made many years ago. As behavior analysts know, individualized operant and classical conditioning histories are critical in the development of behavioral patterns and characteristics.

For the purposes of heritability estimates, genes *and* environments can be directly controlled only for plants and some nonhuman animals, and even then, these efforts often fail. In a *Science* article (Crabbe, Wahlsten, & Dudek, 1999), for example, mice from the same genetic strains were raised in different laboratories under environments rigorously controlled to be as similar as possible. On a number of behavioral tests, however, different laboratories found different results for the same genetic strain, differences sometimes bigger across laboratories than across strains. (The short-term, less-than-precise nature of the tests, such as open field and maze, as contrasted to longer term behavior-analytic operant and respondent assays, makes these results less surprising;[3] also see Francis, Szegda, Campbell, Martin, & Insel, 2003.)

For humans, bombarded by rich and varied experiences every day, many of the environmental factors

[3] Encouragingly, Crabbe et al. conclude by noting that "increased communication and collaboration between the molecular biologists creating mutations and behavioral scientists interested in the psychological aspects of behavioral testing will benefit both groups" (p. 1672).

cannot even be measured, let alone controlled. Scientists do not even know which ones to attempt to measure. For example, only recently have data been collected suggesting the critical importance for language and cognitive development of the sheer volume of speech addressed to toddlers. In Hart and Risley's (1995) longitudinal study following 10-month-old babies until they were 3 years old, analyses of monthly in-home naturalistic samples demonstrated this as one of several critical variables having high correlations with outcome measures such as IQ, a major focus of behavior genetics researchers. These are a handful of the many environmental factors known to affect children selectively even in ostensibly similar environments. In this regard, Hart and Risley found marked differences among working-class families in their critical variables and in the corresponding later outcomes. Several experimental studies have suggested that intense interventions providing the extra stimulation can have significant longer term benefits, including increases in IQ (e.g., the Milwaukee Project, Garber, 1988). No heritability studies have taken these variables into account.

The fact that environmental features can covary with genes adds another complication, illustrated by the classic example of pellagra. This disease of malnutrition was once claimed to be genetic because it appeared to run selectively in families: Family members of those with pellagra were more likely to have the disease than nonfamily members. Heritability estimates would probably have been fairly high. High heritability can of course mean that a genetic abnormality is important, as in the case of PKU, but in this case it did not. Instead, socioeconomic status, naturally correlated with degree of genetic relatedness, proved to be the key: Those who were poor were simply failing to obtain adequate supplies of Vitamin B3. The point is that an environmental factor unknown at the time was confounded with genetic relatedness. A more recent example is the prion-caused disease of kuru, which was initially thought to be genetic (see Jablonka & Lamb, 2005).

Cultural factors are often directly correlated with genetic variation, with sex and race as classic examples (although such genetic differences are small, e.g., Morange, 2001). Skin color continues to affect the way that people are treated, for example.[4] As Moore points out, "In the language of behavior genetics, genes that contribute to skin color differences could fully 'account for' racial IQ differences, even if these genes influence IQ *only* via racist attitudes and behaviors present in our society" (p. 47).

Heritability estimates are based in effect on the averaging of environmental factors. A factor like racism, which is known to correlate with genes, must be statistically accounted for, to the extent possible (e.g., through analysis of covariance; see, e.g., Hays, 1994, and Nunnally & Bernstein, 1994, on the necessary restrictive assumptions). Behavior analysts are in an especially good position to recognize the difficulties with this approach. Without knowledge of the actual causal relations, the effort to control for the many confounding variables statistically is limited in its effectiveness (see Block, 1995; Moore, p. 251). As Mayeux (2005) put it, even in the context of genetic diseases, "heritability estimates do not effectively separate shared genetic from shared environmental influences and cannot effectively apportion the degree of gene-environment interaction" (p. 1405). The construct does have a few valid

[4] Note that skin color can be changed through nongenetic means. The white author of *Black Like Me* changed his skin color to experience life as a black man in the South, resulting in a powerful and influential work (Griffin, 1961).

applications, as Moore notes, but Block (1995) concluded that heritability as used in the IQ controversy was "a lousy scientific concept" (p. 121; see also Farber, 1981; Layzer, 1974).

Genetic Determinism and the Twin Studies

After a period of renewed debate instigated by Herrnstein and Murray's (1994) *The Bell Curve* (to which Block, 1995, was responding), a consensus that this is the case may now have been achieved by those in this field (e.g., Downes, 2004). Other developments have converged, such as the acceptance of the well-documented steady increase in IQ in many developed nations over each succeeding decade (see Moore on the Flynn effect). The consequences of the nature–nurture misunderstandings have been and continue to be serious, though. Genetic determinism, itself problematic, has sometimes been accompanied by an implicit or explicit assumption that environmental interventions are futile or limited in effectiveness. Moore describes the effects of such views on social policies, cultural beliefs, and individual actions. And he does not shrink from the larger political implications. He notes, for example, that genetic determinism for intelligence could be and sometimes has been taken to imply a lesser need for access by all to quality education.

> Given the fact that it is simply impossible to identify people who are *genetically* unable to benefit from access to social resources like quality education and nutrition, it seems incumbent upon democratic societies to distribute these resources equitably. The fact that genetic information alone will *never* be able to specify which people would benefit *most* (or least) from access to these resources merely serves to reinforce this exigency. (p. 215; also see Holtzmann, 2002)

As noted above, heritability estimates for so-called genetic diseases must be both performed and interpreted with considerable caution. The provenance of a disorder like autism is of great concern, and heritability estimates are usually high (e.g., Ronald et al., 2006). However, autism and autism spectrum disorders have apparently been increasing in incidence (although some consider the increase to be illusory). Their causation is still unknown despite years of effort, but research proceeds, and a specific gene abnormality was recently suggested as a predisposing factor (e.g., Campbell et al., 2006). Understanding causation requires learning about multiple genes and environmental factors and their interactions, all this as a function of the range of variability in these factors—clearly a formidable task (see Jablonka & Lamb, 2005, for a detailed example). Thus, shared genes and shared environments can still be extremely difficult or even impossible to disentangle with current techniques. And because of incomplete penetrance and variable expressivity, even in the case of diseases like PKU that are associated with a single gene, sometimes only one identical twin manifests the disorder.

For all these reasons, the genetic determinism sometimes drawn from the twin studies is an obvious target, and Moore's critical analysis makes enjoyable reading. Genetic determination has been suggested for very unlikely traits indeed. The occurrence of coincidences is especially beloved by the mass media: If identical twins raised separately both love woodworking, tell "knock-knock" jokes, and marry men with the same first names, surely these characteristics must be "genetic"? Genes code for proteins, not first names, but confirmation bias is rampant, and dissimilarities can go unexamined. As Moore discusses, such coincidences are due mainly to growing up in the same era, and usually in the same social class and the same or similar neighborhood, as has been documented. As a result, comparable unrelated individuals can also share a sur-

prising number of similarities. (And such environmentally influenced similarities that are not explicitly accounted for statistically can and do serve to inflate the heritability estimate.) On top of this factor, the effects of similar appearance can be dismayingly large,[5] especially important for comparing fraternal and identical twins. Finally, according to Moore, about one third of identical twins (but no fraternal twins) share a chorion, a membrane that is part of the placenta, and hence experience more similar prenatal environments. Some researchers have documented observable effects of this variable.

Similarities across any two people, related or not, are due to genes and environment working together in their complex, interacting ways. Heritability percentages are problematic even when applied to the groups from which they are drawn. Why, then, does it seem reasonable to many that a trait might be 70% genetic and 30% environmental in an individual? As Moore points out, it is eminently intuitive that some traits, like the shape of a nose, seem to be less influenced by environmental factors, whereas others, like hair style, seem more environmentally determined. And of course, in a limited sense, that could be taken to be true (see the previous discussion of ''genetically determined'' and ''environmentally determined''). But the many caveats are very important.

DEVELOPMENT AND EVOLUTION

Development, Environmental Factors, and ''Instincts''

If a behavior occurs despite a large range of individual and environmental variability, the tendency has indeed been to consider it ''instinctive'' or ''innate''; perhaps genetically determined. Adding yet more caveats, Moore summarizes the pioneering work of developmental psychobiologist Gilbert Gottlieb on the provenance of a species-typical behavior like imprinting, which used to be thought of in this way. Gottlieb's research with duckling imprinting showed that nonobvious experiential factors could be critical to the development of innate behaviors such as the unlearned preference for the species-typical maternal call. In one species, ducklings had to hear their own or siblings' contact calls prenatally in order to develop the normal preference, even though these calls bore no resemblance to the maternal call. In another species, perinatal experience hearing siblings' alarm calls was essential. Thus, the normal developmental canalization toward species-typical preference included not only genes, physiological contributors, and other expected variables, but entirely unexpected environmental factors as well. Gottlieb discovered that, as a result, preferences for other species' calls could readily be induced by environmental manipulations (Gottlieb, 1997; see Schneider, 2003, for a review and commentary).

Genes and environment always work together to produce any trait, and examples like Gottlieb's show how ''*all* traits are acquired'' (Moore, p. 203). A critical recognition is

> the understanding that traits that seem impervious to experience are no more ''genetic'' than are traits that seem ''open'' to such influence. The extent to which experiences influence a trait's development reflects a variety of factors …, but it does not reflect the

[5] Identical twins do tend to look similar, especially when raised in similar environments, but this is not necessarily the case. They can be very different in appearance as well as in other characteristics; even cloned animals can look dissimilar (see Moore). The degree of environmental similarity is an obvious factor. In corroboration, Fraga et al. (2005) found that identical twins developed increasingly different epigenetic DNA patterns as they aged, especially those spending less time together or having different medical histories (also see Gottlieb, 1997).

extent to which genes control the trait's development. (Moore, p. 185)

Farewell (again) to the percentage game.

As Moore points out, the detection of such nonobvious contributors requires special care. Mother rats' licking of male preweanlings has been shown to be essential for the later development of normal sexual behavior (C. Moore, 2003). However, separating the pups from their mother after weaning, raising them in social isolation, and observing normal sexual behavior might be taken to suggest that the environment is unimportant, which is clearly far from the case. Many such examples of nonobvious environmental contributors are now known to exist (see, e.g., Lickliter & Honeycutt, 2003).

Experience is critical for development in myriad ways, and Moore notes research showing that corresponding brain plasticity is now known to be higher throughout the lifespan than had been thought. The fantastic chimeras created by embryologists who combine parts of different creatures have demonstrated how the environment, not the genes, determines which cells become parts of what organs, and just how plastic that process is. "We are standing and walking with parts of our body which we could have used for thinking if they had been developed in another position in the embryo" (Spemann, quoted in Moore, p. 87).

Moore, an infancy researcher himself, focuses especially on perinatal development, the source of an explosion of news over the past few decades. One phenomenon is fetal programming, a lifelong predisposition to obesity caused by poor maternal nutrition at a particular prenatal stage. Of special interest to behavior analysts, Spear and his colleagues have shown that placental or mammary exposure to ethanol (at levels far below those for fetal alcohol syndrome) establishes it as a reinforcer later, and can perhaps contribute to alcoholism (e.g., Spear & Molina, 2005; this particular research line is not cited in *The Dependent Gene*). Here again, confusion can arise over genetic and nongenetic familial inheritance patterns.

Behavioral Inheritance

Developmental work has complemented behavioral work in documenting nongenetic inheritance mechanisms in addition to the more molecular epigenetic ones discussed previously. For example, it has long been known in humans and other mammals that acquired immunity can be transmitted nongenetically, through breast milk and the placenta. Similarly, when a female Mongolian gerbil embryo is positioned near brothers rather than sisters, she is exposed to more testosterone, and, like her male siblings, is likely to be licked more than female-positioned females (Clark, Bone, & Galef, 1989; Clark, Karpluk, & Galef, 1993). Later behavioral effects include greater aggression and the ability to hold larger territories. Such female gerbils tend in turn to have male-dominated litters, so their daughters show the same patterns, thus providing another illustration of nongenetic inheritance. Behavioral mechanisms are involved, and the extra licking provides an excellent example. Further, as discussed previously, similar extra licking of male pups by rat mothers was demonstrated to be critical for later male sexual behavior. This behavior has been shown to be caused by testosterone or associated hormones in the male rat pups' urine, which act as a reinforcer for the mothers' licking (C. Moore, 1982, 1995).

Cross-fostering studies, in which young of one genetic strain are reared by mothers of a different strain, are especially useful in studies of gene–environment inheritance relations. Cierpial and McCarty (1987) used this

approach to show that rats of the spontaneously hypertensive (SHR) genetic strain do not show the SHR behavior pattern if raised by non-SHR mothers (see also Ressler, 1966, discussed by Moore; C. Moore, Wong, Daum, & Leclair, 1997; Suomi, 1999). Integral once again were behavioral mechanisms similar in some ways to the differential maternal handling discovered by C. Moore (Cierpial, Murphy, & McCarty, 1990).

Operant behavior comes even more to the forefront in the social learning that is an obvious behavioral inheritance mechanism. Berman (1990) noted likely operant involvement in the maternal parenting styles that tend to be passed down from mother to daughter for generations in rhesus monkeys (see Fairbanks, 1996, and Suomi, 1999, for related research). For example, access to an infant sibling is reinforcing for most females, and maternal rejections of the infant can be discriminative stimuli signaling an opportunity for access. Attention to the mother's parenting of the sibling is sometimes reinforced by access to the mother as well. Berman suggests that such stimulus control facilitates learning of a parenting style through imitation (which of course involves operants; see, e.g., Chase & Masia, 1997). Observational learning is also critical for the transmission of foraging techniques. An impressive variety of such behavioral inheritance mechanisms across the animal kingdom is documented in *Animal Traditions: Behavioural Inheritance in Evolution* (Avital & Jablonka, 2000), a work in which operant involvement is explicitly recognized.

The evolutionary implications are significant. Moore only footnotes the Baldwin effect—the idea tracing back to Lamarck and Darwin that behavior can initiate evolutionary change (see Avital & Jablonka, 2000; C. Moore, 2003; Schneider, 2003). However, he emphasizes two key associated insights. First, environments are passed along rather like genes (and the essential cytoplasm containing the genes):

> To the extent that we cannot help but develop in environments that are similar in important ways to the environments in which our parents developed, the legacy we receive from our parents includes both our genes and aspects of our developmental environments. (Moore, p. 174)

Evolutionarily speaking, both genes and critical features of environments are and must be reasonably stable across generations. Second, as Moore points out, natural selection does not act directly on genes, but on phenotypes. Phenotypes are produced and modified by both genes and environments, and behavior principles have an important role. Evolution might even be considered to proceed by lasting phenotypic changes regardless of whether there is an accompanying change in the genome, a controversial proposal made by Gottlieb (Moore, p. 201). These lines of thought are at the heart of the integrative, empirically based approach to nature–nurture relations known as developmental systems theory.

Developmental Systems Theory

The Dependent Gene is one of the first trade books on developmental systems theory, which encompasses all the research areas bearing on nature–nurture relations. Behavior analysis is eminently consistent with this approach, one that makes the role of environmental factors like behavior principles explicit. The very title of a recent edited work in this tradition is significant: *Cycles of Contingency* (Oyama, Griffiths, & Gray, 2001). Moore's book provides an excellent introduction. A more technical work of epic scope is Jablonka and Lamb's (2005) *Evolution in Four Dimensions*: *Genetic, Epigenetic, Behavioral, and Symbolic Variation in the History of Life*. Other notable recent books that can reasonably be grouped under the de-

velopmental systems rubric include Avital and Jablonka (2000), Blumberg (2005), Gottlieb (1997), Oyama (2000), and West-Eberhard (2003). For behavior-analytic reviews, see Midgley and Morris (1992) and Schneider (2003).

The Dependent Gene is well documented with ample footnotes. However, from a behavior-analytic point of view, the lack of coverage of operant and respondent involvement in nature–nurture relations is disappointing (Avital & Jablonka, 2000, and Jablonka & Lamb, 2005, do better). It is also sad to see the inaccurate ''extreme environmentalist'' characterization of John B. Watson's role in the nature–nurture battle (see Todd & Morris, 1992; this will be corrected if a revised edition is issued). Finally, in Moore's valuable evolutionary discussion of heterochrony (changes in developmental timing), an update on the nature of its role in human evolution may be required (e.g., McNamara, 1997).

Environmental Determinism

Moore's book focuses on dangers of the concept of genetic determinism. Scientists' new power to investigate the complex causation in nature–nurture relations has benefited, of course, from the mapping of the human genome. The resulting tendency to focus on the genes does not *necessarily* lead to less effort to understand the environmental contributors by any means, but it can have that effect. Known genetic involvement even of a single-gene single-trait type clearly does not mean that a particular outcome is unavoidable (any more so than in cases referred to as ''environmentally determined''). Such examples can still be amenable to environmental and behavioral interventions, and the monogenic ''genetic'' disease PKU is a good case in point. Eliminating the indigestible amino acid from the diet currently provides the best treatment. The particulars of each problem determine how best it can be handled, so, in the future, some problems thought of as environmentally determined may be best dealt with through gene therapies. (For now, those therapies appear to remain distant possibilities.)

Moore also notes that, although it is inherently less likely to lead to the stuck-with-it do-nothing outcome that has sometimes resulted from genetic determinism, environmental determinism is problematic too. After all, environmental interventions operate on organisms built in part by genes, and they continue to be affected by genes through gene products. Even features that seem largely controlled by environmental factors for almost everyone are influenced by genes, and can be very different given enough of a change in the genome. An obvious example for behavior analysts is learning, in the case of PKU or Down syndrome, with their documented genetic contributions. But more subtle examples exist too, and behavioral interventions may sometimes fail to work because of unrecognized genetic factors (see, e.g., Manuck, Flory, Ferrell, & Muldoon, 2004; Suomi, 2002, 2003). Knowledge of such genetic involvement would be very helpful even without the existence of gene therapies. If they were to exist, the known presence in an individual of genetic predispositions for alcoholism or autism, for example, means that behavioral and other environmental countermeasures could be targeted at an early age. The presence of interactions means that the predispositions might be problematic only in particular environments to begin with.

CONCLUSION

The 21st century brings a revolution in our understanding of nature–nurture relations, one that clearly goes far beyond the mapping of the human genome. As *The Dependent*

Gene documents, genes and environmental factors interact at all levels in very complex ways. The more dissemination of this spectrum of findings, the better for fields like behavior analysis that are focused on behavior–environment principles that do not always get the same respect as genetics. Ironically, many geneticists do recognize the important role of the environment (e.g., Moore; Morange, 2001), although that message has not always been well publicized.

Similarly, behavior analysts have always recognized the importance of genetic involvement in the phenomena they study (and now the practical implications are growing). But that fact has not always been acknowledged either: As Morris, Lazo, and Smith (2004) documented, although B. F. Skinner wrote amply about biological, genetic, and evolutionary involvement in behavior, he was and continues to be frequently accused of neglect. Behavior analysts can be proactive by talking knowledgeably about their science's relation to the larger life sciences—and the pivotal role of the behavior processes they study and apply. Awareness of the nature–nurture relations described in *The Dependent Gene* can provide support as well as illumination.

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